Modern Concepts of Cardiovascular Disease

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THE ACUTE AND CHRONIC PHASES OF CORONARY ATHEROSCLEROSIS

Coronary atherosclerosis has its acute and chronic phases. In the following discussion these phases will be referred to as acute and chronic coronary disease.

Acute Coronary Disease

The acute phase of coronary disease may be regarded as that phase which is associated with any new clinical picture resulting from a sudden anatomical decrease in the coronary volume produced by partial or complete occlusion. This occlusion may or may not be followed by infarction.

This sudden change in the overall coronary capacity may announce itself as (1) simple angina pectoris, (2) an aggravation of preexisting angina, (3) angina decubitus, or (4) a single attack of prolonged cardiac pain with or without infarction. Regardless of the manner in which the acute phase appears, it may be accompanied by coronary thrombosis (clinically undiagnosable) or it may be followed shortly by myocardial infarction (usually easily diagnosable).

It is important to recognize that the onset of even simple angina pectoris represents an acute change in the coronary status. On one day some customary activity does not produce pain; on another day it does. Something must have happened in the coronary vessels in the intervening day. It is probable that there is a period of hours to days and perhaps even to weeks after the onset of the angina pectoris when there is a threat of serious complications from the episode which produced the onset of the angina pectoris. Such complications may cause sudden death or cardiac infarction. After a few months the change which produced the angina pectoris may be said to have become stabilized so that any subsequent important change, if not explainable by extracoronary factors, must be considered a new coronary episode. Angina pectoris therefore may have its acute and chronic phases.

In general it may be said that the diagnosis of acute coronary disease may be made whenever there is a new constellation of coronary symptoms that cannot readily be explained by extracoronary factors such as cold, unusual exertion, hyperthyroidism, anemia, etc. An attack of prolonged cardiac pain, with or without infarction, is generally recognized as acute coronary disease. It seems worth while to extend the concept of acute coronary disease to include the other categories referred to above.

According to this view there is some question as to whether one should think of a short preliminary period of angina pectoris as premonitory evidence of myocardial infarction. The myocardial infarction is perhaps more properly viewed as one of the complications of acute angina pectoris. For while angina pectoris may be followed, shortly after its onset, by myocardial infarction (because of the recent narrowing or occlusion) it is perhaps more likely to continue to the chronic state which is so generally recognized as classical angina pectoris or it may in fact, after a variable

ANNUAL DINNER

The Annual Dinner of the American Heart Association will be held in the Vernon Room, Haddon Hall, Atlantic City, New Jersey, 7:30 P.M., Saturday, June 9, 1951. Members are invited to bring their family and friends. Reservations at $7.00 each may be made at the time of registration in Atlantic City or by writing directly to the American Heart Association.

period, disappear. This concept would also suggest that the myocardial infarction which follows the sudden worsening of a pre-existing angina pectoris is likewise a complication of a new episode.

One form of acute coronary disease deserves particular mention, namely, angina decubitus. There is certain clinical merit to the designation of angina decubitus as acute coronary disease for at least several weeks after its onset. In the first place, it indicates that one is dealing with a period of transition. Secondly, it keeps one on the alert for imminent and serious complications. Thirdly, it makes one aware of the real possibility of more or less spontaneous improvement; the knowledge that in time great improvement may occur spontaneously is a source of hope which is of great help to the physician and to his patient in that trying period of almost complete invalidism. It is the failure to recognize the frequency of spontaneous and considerable improvement in acute coronary disease with angina decubitus which has occasionally led to the misinterpretation of striking benefits resulting from various therapeutic efforts.

Except in the case of myocardial infarction there may be no electrocardiographic changes accompanying the onset of an acute coronary episode. Or the changes may be minimal and transient. However, there may follow within a matter of days or weeks significant electrocardiographic changes, this despite the absence of clinical evidence to point to such changes. It is for this reason that frequent electrocardiographic tracings should be obtained in the first few weeks after the onset of an acute coronary episode, even though there is no clinical evidence to suggest an infarct. One can assume on clinical grounds that there has been an episode of narrowing or occlusion, and one can determine electrocardiographically whether or not this episode has resulted in transient anoxic myocardial change, subendocardial necrosis, or myocardial infarction.

An increase in the severity of angina pectoris may of course be due to extracoronary factors which are generally obvious, such as emotion, work, temperature, weight gain, increased smoking, hyperthyroidism, and anemia. An aggravation of angina pectoris due to extracoronary factors is likely to come on not quite so abruptly as the worsening due to coronary occlusion, partial or complete. The reasons are clear. Hence the history alone may indicate whether the increased severity of angina pectoris was brought about by an anatomical change in the coronary vessels or by factors outside the coronary arteries. It is important especially to bear in mind hyperthyroidism and anemia as extracoronary causes for an aggravation of anginal symptoms because these states are easily overlooked and because they are generally correctible.

Chronic Coronary Disease

Having passed through the period designated as acute, the coronary episode reaches a chronic state. This state may be characterized by (1) angina pectoris, (2) congestive heart failure, (3) angina decubitus, (4) no recognizable coronary symptoms.

When angina pectoris has become sufficiently well established it is the most easily recognizable form of chronic coronary disease. In the chronic state angina pectoris is remarkably constant under fixed conditions, as indicated by exercise tolerance tests. To be sure, the severity of the angina pectoris may vary considerably as a result of extracoronary factors as indicated above. However, the underlying impairment of the coronary vessels, the chronic coronary disease, remains unchanged.

One of the chronic states which may result from an acute coronary episode is congestive heart failure. The preceding acute episode may even have passed unnoticed, as is often the case. However, the general clinical situation, often with helpful electrocardiographic changes, may point to an acute coronary incident which might have preceded the onset of the congestive heart failure. It is important that this diagnosis be made if possible, since a patient with congestive heart failure as a manifestation of chronic coronary disease is subject to subsequent coronary occlusive episodes, as is any other patient with chronic coronary disease. His prognosis therefore is not determined wholly by the fact of the congestive failure but by the fact of the coronary history as well.

Angina decubitus may persist long enough to qualify for inclusion in the chronic state. This, however, is unusual.

Once there has been recovery from an acute coronary episode it may be assumed that the chronic state of coronary disease will evolve. This is true whether or not there is persistent cardiac pain or congestive heart failure. For even if there are no symptoms following an acute episode, the diagnosis of chronic coronary disease should be made if for no other reason than to force a recognition of the fact that a patient in this state is subject to further coronary episodes, just as is the patient with chronic angina pectoris. The coronary disease with its anatomic changes is present even though there may be no symptoms of the coronary disease.

Asymptomatic chronic coronary disease may also be present when there has been no previous acute episode. It is known from autopsy studies that a large proportion of apparently normal males beyond middle age have significant coronary atherosclerosis. The presence of this atherosclerosis becomes evident clinically only by symptoms or electrocardiographic changes. The disease is present, however, whether or not there are symptoms or electrocardiographic changes. This form of chronic coronary disease is undetectable in the living patient. Yet such a patient is subject to an acute coronary disturbance just as is the patient with chronic angina pectoris. If it is true that at least fifty per cent of apparently normal males beyond forty-five years of age have coronary disease, then at least fifty per cent of the normal male population beyond forty-five years of age should be viewed, from the standpoint both of treatment and prognosis, in terms of patients with chronic coronary disease. Placing a large part of the male population beyond middle age in the chronic coronary disease category may ultimately influence significantly our point of view not only toward this group but also toward clinically recognizable chronic disease as well. For if patients with recognizable chronic coronary disease are not essentially different from, let us say, half of the male population beyond middle age, they are more nearly "normal' than we have been inclined to believe in the past. And, contrariwise, perhaps the "normal" group are not nearly so normal as we have been inclined to believe. Perhaps we shall come to be less surprised when patients with recognizable chronic coronary disease live long useful lives as we become less surprised when apparently normal people develop acute coronary episodes.

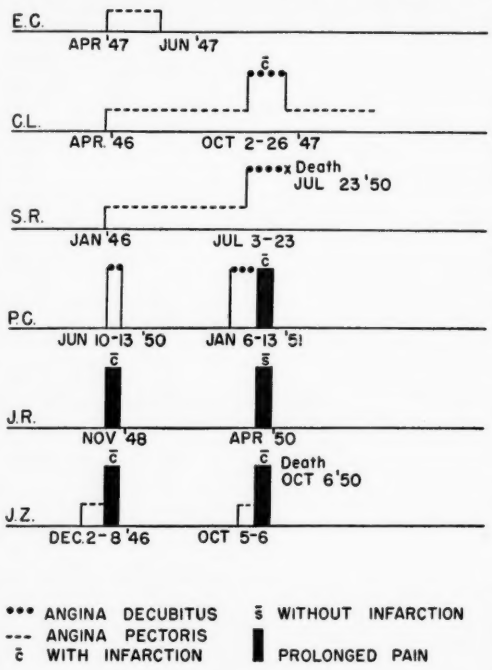

Figure 1

General Discussion

Coronary disease with pain does not usually proceed in a smoothly or even irregularly progressive manner from mild to moderate to severe angina pectoris as one might expect were the pain simply a reflection of a gradual narrowing of the coronary bed. If such a course were common one would anticipate that most patients with coronary disease and pain would finally become bedridden and die in such a terminal state. Actually most patients with coronary disease die while relatively active. It may be that the gradual narrowing of larger coronary vessels favors the development of collateral channels since time is allowed for the slow transition from one channel to another. Under such circumstances, the gradual occlusion of coronary vessels might well produce little or no clinical evidence of increasing coronary insufficiency for the reason that there is in fact no functionally significant overall decrease in the coronary bed. In any event, clinically one does not commonly observe a progression of increasing angina pectoris. Rather, excluding the influence of extracoronary factors, the course of coronary disease with cardiac pain is usually characterized by one or more new episodes, and each new episode may appear in a different guise. It is likely that each new episode results from a new narrowing or occlusion. This course is illustrated by the diagrammatic representation of clinical cases, as seen in Figure 1. The changes in the severity of the coronary disease due to extracoronary factors are excluded in these schematic illustrations, since such changes are unimportant so far as the underlying disease is concerned and since they would obscure the basic changes in the natural history of the coronary disease. In the past there has perhaps been insufficient emphasis on the necessity for clearly separating the influence of the extra- from the intracoronary factors in modifying the course of the disease. We have tended to think too much in terms of whether the angina pectoris was better or worse rather than whether the disease itself had changed.

The categories of angina pectoris (in its chronic form) and myocardial infarction have been sufficiently long recognized as spe-

cific syndromes so that these diagnoses are easily made, their management satisfactorily established, and their natural history fairly predictable. The same cannot be said at present for any of the other categories included in the acute and chronic forms of coronary disease. When these categories are separately recognized it will be possible to evaluate therapy and to judge prognosis more satisfactorily than is now the case. Thus, for example, whether the angina pectoris is in the acute or chronic phase will affect the treatment probably, and the prognosis surely. A separate clinical evaluation of therapy and a separate study of prognosis are necessary for the separate stages of the angina pectoris. Should a patient with acute angina pectoris be put to bed and for how long? Should he be given anticoagulants? Need a patient with chronic angina pectoris be greatly restricted in his mode of life? Should the question as to rest or exercise in angina pectoris be viewed in terms of whether the angina pectoris is acute or chronic? What is the relative seriousness of acute and chronic angina pectoris? These and other questions need to be answered not only for angina pectoris but also for the separate stages of the various manifestations of coronary disease. This is a fruitful field for clinical research.

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The American Heart Association wishes to call to the attention of its members that it has received a second grant from Wyeth, Inc., Philadelphia, Pennsylvania, to the Publication Fund of the Association. This grant permits the use of color in illustrations in our official publication—CIRCULATION, the Journal of the American Heart Association.

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